# VASCULAR DISTURBANCES ASSOCIATED WITH FRACTURES*

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THE vascular disturbances associated with fractures constitute a fascinating group of problems. In some instances these disturbances are fraught with grave consequences, so that their early recognition and the institution of proper treatment are of paramount importance. In wounds of the extremities involving a major artery, DeBakey and Simeone[1] in World War II, found an incidence of amputation of 50 per cent. In the Korean conflict the application of newer blood vessel techniques to the problem of major arterial repairs resulted in a reduction in the amputation rate to 17 to 22 per cent.[2,3] While the problem of arterial damage is encountered less commonly in civilian practice, its consequences are such as to require us to be alert to any new techniques which may save limbs and yield better functional results. With this in mind let us consider first the immediate disturbances in arterial supply to the injured extremity and then follow with a short review of some of the remote changes.

The early troubles are of three types: 1) injury or division of the main artery of supply to the affected extremity; 2) compression of the main artery by hematoma or occlusion by angulation; and 3) severe vasospasm simulating occlusion. The signs and symptoms of impaired blood supply of the extremity are well known and consist of pain, coldness, numbness, pallor, empty veins, absent pulses, and with the passage of time, dependent lividity. Oscillometric readings are markedly diminished or absent distal to the occlusion. Naturally, the general condition of the patient affects these signs and symptoms. For example, a small degree of obliteration may be markedly accentuated by a shock-like state of the patient. Further, the pre-existence of any degree of obliterative disease may set the stage for obstruction

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and a check of the vessels in the other extremities may yield information regarding the initial state of the artery.

If it is apparent that the blood supply to the extremity is impaired, it is desirable to try to evaluate the role played by spasm. This is best done by some form of nerve block designed to interrupt the sympathetic fibers to the extremity. If the result is a very dramatic improvement in the appearance of the extremity following block, then obviously spasm is playing a dominant role and continued observation is justifiable with further efforts directed at the release of spasm. If, however, the result is slight, or if there is considerable hematoma or other reason to believe there is compression of or actual injury to the artery, exploration of the vessel should be considered. Before undertaking such a procedure, however, it is wise to have some idea of the consequences of sudden occlusion of major vessels untreated by surgery. According to four different authors, Wolff,[4] Makins,[5] Heidrich,[6] and Haimovici,[7] the incidence of gangrene ranges as follows: axillary artery 10-15 per cent, brachial artery 3-4 per cent, aorta 55-100 per cent, common iliac artery 40-100 per cent, external iliac artery 11-17 per cent; common femoral artery 22-26 per cent; superficial femoral artery 10-14 per cent, popliteal artery 15-37 per cent. With some understanding then, of the consequences of occlusion at different levels and bearing in mind that most explorations may be carried out under local anesthesia, where the patient's general condition is poor, and also bearing in mind that the extremity vessels are fairly readily accessible, the presence of perfectly obvious impairment of arterial flow requires exploration at the site of injury. Adequate blood for transfusion should be available, of course, for any blood vessel operation.

If the signs indicate that the vessel may be ruptured or if there is a large hematoma present, particularly if the latter pulsates, one should adhere to the fundamental vascular principle of obtaining careful control of the main artery above and below the area of hematoma before dissecting into it. Otherwise, with the first few cuts into the fascial compartments containing the hematoma, the release of tamponade pressure may be followed by a sudden gush of blood and considerable ensuing hemorrhage. The latter results in frantic and awkward attempts to control the bleeding and may be associated with undue blood loss. If, on exploration, the vessel is ruptured or badly trauma-

tized, the injured segment should be excised and either rejoined with end-to-end anastomosis or replaced with a prosthesis. The latter may be of three types, either 1) autogenous vein, 2) preserved homograft, or 3) a plastic cloth tube of suitable material such as Vinyon-N or Dacron, both of which are now under investigation as vessel replacements. If the injury is slight, lateral repair may be attempted provided it does not appreciably reduce the lumen of the vessel.

When neither a homograft nor plastic cloth prosthesis is available, then one will be forced to consider the use of an autogenous vein. Usually, a suitable vein can be found of proper length. It should be slightly smaller in diameter than the artery it is to replace since veins distend somewhat when filled with blood under arterial pressure. If the graft selected is already of the same diameter then it will be larger than the artery when carrying arterial blood, and form a fusiform dilatation which tends to propagate into an actual aneurysm. The vein segment should also be valve free, if possible, in order to prevent eddy currents which encourage the settling out of thrombi. The disadvantages of veins as grafts are aneurysm formation and thrombotic occlusion, particularly when long lengths are used.

If the blood vessel to be replaced crosses a joint or flexion crease, many have thought that the prosthesis of choice is a preserved homograft. Such arteries have already been shown to function nicely as replacements.[8-11] One barrier to the use of homografts lies in their scarcity, unless one has a large vessel-bank available with a wide variety of arteries. A second difficulty is that a few late blowouts have been reported in preserved homografts. A third objection is that sclerosis has appeared in the walls of such grafts two to four years after resection.[8]

Plastic cloth sewn into tubes of the proper diameter and length have been used by us to replace segments of the femoral and popliteal arteries. These cloths should probably not be used in vessels much below 4 mm. in diameter. We have had some experience with a cloth known as Vinyon-N[12] which has the property of retaining its tensile strength and which is markedly inert. It functions as a strut through the interstices of which fibroblasts grow to the inner lining surface where they assume a flat shape indistinguishable from endothelium. One objection to Vinyon-N is that the cloth now available has a high porosity through which a rather extensive blood loss occurs at the time of release

of the hemostatic clamps. Various other cloths have been employed in other parts of the country[13, 14] and efforts are now being made to study the different fabrics of Orlon, Dacron, Nylon, Vinyon-N and others in an orderly fashion, using comparable filament size, weave, twist, weight, and porosity. At the moment Dacron and Nylon cloths of low porosity appear to be suitable materials for blood vessel replacement.

If one should escape the immediate arterial difficulties, later complications are still a possibility. These consist in secondary hemorrhage, aneurysm formation, arteriovenous fistula, and so-called post-traumatic reflex sympathetic dystrophy. Any of us concerned with any volume of traumatic surgery has witnessed the frightful spectacle of massive secondary hemorrhage. This is commonly related to necrotizing infection and in this respect it is worth while calling attention to the gas forming organisms as possessing some potential for tissue destruction. Not infrequently life is saved in such cases by quick thinking ward attendants applying pressure at the proper point until the surgeon can be summoned. If infection is present, as it usually is, consideration of blood vessel replacement is out of the question and one is only concerned with controlling the hemorrhage by ligation of the vessel. This requires no special comment. Occasionally, however, the secondary hemorrhage may result from the rupture of an undetected or false aneurysm, secondary to the initial trauma. If this is the case, then replacement of the blood vessel by one of the techniques previously described is in order. Such an aneurysm might be suspected before rupture, if a pulsating mass is palpated in the region of injury. Should there be any question as to the diagnosis, this may be elucidated further by performing arteriography, providing the patient is not so immobilized in plaster as to make such an attempt out of the question.

An arteriovenous fistula may announce its presence by the appearance of dilated and tortuous veins with stasis pigmentation, dermatitis or ulceration distally, the presence of a thrill, palpable over the area of the fistula and the auscultation of a to-and-fro murmur, characteristic of such fistulae. Here again, if there is any question of the diagnosis, an arteriogram can be performed and may be of tremendous help. The treatment is surgical and depends upon the site of the fistula. Four point control must be obtained before any direct attack is attempted on the fistula itself. This means control of the artery and the vein

involved both proximal and distal to the fistula. Once this is done it may be possible to sacrifice the vein and to repair the defect in the artery with an over-and-over stitch. On the other hand, arterial repair may not be possible due to the size and shape of the lesion and it may be necessary to resect the fistula and replace the arterial defect with a prosthesis, as before mentioned.

Now, we come finally to a large group of disorders which may all be classified under the heading of post-traumatic reflex sympathetic dystrophy. When such autonomic disturbances are associated with severe pain, edema, skin changes, tenderness and hyperesthesia, they are called causalgias or the Weir-Mitchell syndrome. When they are associated with marked vasospasm, blanching and sweating of the extremity, they are called reflex, post-traumatic Raynaud's phenomena. And when they are associated with vasomotor disturbances and certain osteolytic changes in small bones they are called Sudeck's atrophy. In whatever form these disturbances present themselves, the underlying dysfunction is an autonomic nervous one. Here, it is worth while pointing out that the picture is not always a consistent one, but may be phasic. For example, a case of Sudeck's atrophy may, in the early period, present a picture of an erythromelalgia-like state with warm digits and a burning type of pain; later, this may be followed by a vasospastic picture with coldness, blanching and sweating. To me it seems less important to differentiate the different types of dystrophy than to recognize their existence early and to study their progression. Certainly, in those associated with causalgia type pain, it is well to interrupt the pattern early before the entire consciousness of the patient becomes fixed upon the pattern of pain. A regional sympathetic nerve block will quickly tell whether or not sympathectomy will bring relief, and if the causalgia is severe, a sympathectomy should be accomplished. Mayfield[15] has reported 105 patients with post-traumatic causalgia of the severe type, treated by sympathectomy, following World War II. In his series, all except nine were completely relieved of their pain. Other authors have reported smaller series with less benefit. In my own rather small experience I have observed a tendency for transferral of pain to other sections, even though relief of pain in the affected limb tends to be complete. This is particularly true in compensation cases.

In summary, some of the immediate and late vascular complications of fractures have been outlined. Some techniques of dealing with blood

vessels have been discussed. It is certain that the proper and timely application of such techniques to injured extremities will not only increase the number of limbs that may be saved after trauma, but will also improve the late functional results.

## *REFERENCES*


1. DeBakey, M. E. and Simeone, F. A. Battle injuries of arteries in World War II; analysis of 2,471 cases, *Ann. Surg. 123*:534-79, 1946.
2. Hughes, C. W. The primary repair of wounds of major arteries, *Ann. Surg. 141*:297-303, 1955.
3. Spencer, F. C. and Grewe, R. V. The management of arterial injuries in battle casualties, *Ann. Surg. 141*:304-13, 1955.
4. Wolff, E. Die Häufigkeit der Extremitätennekrose nach Unterbindung grosser Gefässstämme, *Beitr. klin. Chir. 58*:762-807, 1908.
5. Makins, G. Resultats éloignés des opérations portants sur les gros troncs artériels des membres, *Assoc. franç. Chir. Congr. Chir. 31*:387-94, 1922.
6. Heidrich, L. Ueber Ursache und Häufigkeit der Nekrose bei Ligaturen grosser Gefässstämme, *Beitr. klin. Chir. 124*:607-38, 1921.
7. Haimovici, H. Peripheral arterial embolism; study of 330 unselected cases of embolism of the extremities, *Angiology 1*:20-45, 1950.
8. Coleman, C. C., Deterling, R. A., Jr. and Parshley, M. C. Some long-term observations on aortic homografts, *Surgery 37*:64-79, 1955.
9. Gross, R. E., Hurwitt, E. S., Bill, A. H., Jr. and Peirce, E. C. Preliminary observations on the use of human arterial grafts in the treatment of certain cardiovascular defects, *New Engl. J. Med. 239*:578-79, 1948.
10. Swan, H. et al. Arterial homografts; fate of preserved aortic grafts in the dog, *Surg. Gynec. Obstet. 90*:568-79, 1950.
    Miller, H. H. et al. Fate of arterial grafts in small arteries, an experimental study, *ibid. 92*:581-88, 1951.
11. Deterling, R. A., Jr., McAllister, F. F. and Humphreys, G. H., III. The clinical use of aortic homografts, in press.
12. Voorhees, A. B., Jr., Jaretzki, A., III and Blakemore, A. H. Use of tubes constructed from Vinyon "N" cloth in bridging arterial defects; preliminary report, *Ann. Surg. 135*:332-36, 1952.
13. Shumacker, H. B., Jr., Harris, E. J. and Siderys, H. Pliable plastic tubes as aortic substitutes, *Surgery 37*:80-93, 1955.
14. Hufnagel, C. A. Aortic plastic valvular prosthesis, *Bull. Georgetown Univ. med. Cent. 4*:128-30, 1951.
15. Mayfield, F. H. Causalgia, *W. Va. med. J. 43*:201-05, 1947.